Refine Search

Search Results -

Terms	Documents
L4 and (424/450).ccls.	26

Database:
US Pre-Grant Publication Full-Text Database
US Patents Full-Text Database
US OCR Full-Text Database
EPO Abstracts Database
JPO Abstracts Database
Derwent World Patents Index
IBM Technical Disclosure Bulletins

Search: L5

Refine Search

Recall Text Clear Interrupt

Search History

DATE: Friday, March 02, 2007 **Purge Queries** Printable Copy Create Case

Set Name side by side	Query	Hit Count	Set Name result set
DB=PGPB,USPT,USOC,EPAB,JPAB,DWPI,TDBD; PLUR=YES; OP=OR			
L5	L4 and 424/450.ccls.	26	L5
L4	liposome adj5 succin$	53	L4
L3	Liposome adj5 nickel	13	L3
L2	L1 and 424/450.ccls.	35	L2
L1	liposome adj5 biotin	127	L1

END OF SEARCH HISTORY

First Hit Fwd Refs Previous Doc Next Doc Go to Doc#
Generate Collection Print

L2: Entry 51 of 58 File: USPT Jul 30, 1996

DOCUMENT-IDENTIFIER: US 5541232 A
** See image for Certificate of Correction **
TITLE: Treatment of multidrug resistant diseases

Brief Summary Text (6):
A detailed discussion of multidrug resistance can be found in Endicott, et al., "The Biochemistry of P-glycoprotein--Mediated Multidrug Resistance" Ann. Rev. Biochem. (1989) 58, 137-171, incorporated herein by reference. As discussed in the Endicott article, multidrug resistance (MDR) is a unique phenomenon in the study of cellular drug resistance. Cell lines exhibiting this phenotype have been selected for resistance to a single cytotoxic agent, yet they display a broad, unpredictable cross-resistance to a wide variety of unrelated cytotoxic drugs, many of which are used in cancer treatment. The drugs most often involved in MDR are alkaloids or antibiotics of plant or fungal origin, and they include the vinca alkaloids, anthracyclines, epipodophyllotoxins, and dactinomycin. Cross-resistance to alkylating agents such as melphalan, nitrogen mustard, and mitomycin C is occasionally observed. Collateral sensitivity (increased sensitivity) to membrane-active agents such as nonionic detergents, local anesthetics, steroid hormones, and calcium channel blockers often accompanies the development of MDR. The recognition that the emergence of a complex drug resistance phenotype of broad specificity in human tumors could limit successful chemotherapy has provided the impetus to study MDR cell lines as a model for clinical drug resistance.

Previous Doc Next Doc Go to Doc#

First Hit Fwd Refs Previous Doc Next Doc Go to Doc#

Generate Collection Print

L3: Entry 71 of 128 File: USPT Oct 8, 2002

DOCUMENT-IDENTIFIER: US 6461637 B1
TITLE: Method of administering liposomal encapsulated taxane

Brief Summary Text (12):
U.S. Pat. No. 5,648,090 (Rahman et al.) and U.S. Pat. No. 5,424,073 (Rahman et al.) provide a liposomal encapsulated paclitaxel for a method for treating cancer in mammals using such a liposomal-encapsulated paclitaxel, or anti-neoplastic derivative thereof. The '090 and '073 patents disclose a method of modulating multidrug resistance in cancer cells in a mammalian host by administering to the host a pharmaceutical composition of a therapeutically effective number of liposomes which include a liposome-forming material, cardiolipin, and an agent such as paclitaxel, or an antineoplastic derivative of paclitaxel, or a mixture thereof; and a pharmaceutically acceptable excipient.

Previous Doc Next Doc Go to Doc#

L3: Entry 107 of 128 File: USPT Jul 15, 1997

DOCUMENT-IDENTIFIER: US 5648090 A
** See image for Certificate of Correction **
TITLE: Liposome encapsulated toxol and a method of using the same

Detailed Description Text (37):
Our studies demonstrate that liposome encapsulated taxol has significant capacity to overcome multidrug resistance. These studies were performed in HL 60/VCR cells which are promyelocytic human leukemia cells and are derived from parent HL-60 cells and are made resistant to vincristine demonstrating multidrug resistance phenotype. The HL-60 VCR cells were grown in media at 37.degree. C. with 5% CO.

Detailed Description Text (39):
The survival curves of HL-60/VCR cells after exposure to free taxol and liposome encapsulated taxol are presented in FIG. 5. The IC.sub.50 for free taxol in HL-60/VCR is 2.43 .mu.g/ml demonstrating this cell line is 1280 fold resistant than the parent HL-60 cell line. However, the IC.sub.50 for liposomal taxol is only 0.37 .mu.g/ml. This demonstrates that liposomal taxol sensitizes 7 fold the HL-60/VCR as compared to free taxol. Hence, it is apparent that liposome encapsulated taxol modulates drug resistance in the multidrug resistance phonotype which potentially would be a major advantage in a clinical situation where a large population of the patients fail to respond to chemotherapy because of multidrug resistance.

Detailed Description Text (41):
The cellular content of taxol was determined by High Pressure Liquid Chromatography. In brief, the multidrug resistance HL-60/VCR exponential growth phase cells were incubated in 100 mm petri dishes with drug containing medium at 37.degree. C. containing either free taxol or taxol encapsulated in liposomes. The cells were treated for 1 to 4 hours with either of the drug and were then centrifuged and rinsed twice with PBS. The cells were suspended in cold PBS and counted, and centrifuged again. The cell pellets were suspended in 0.5 ml of 2% SDS solution and then sonicated for 5 minutes in a cup-horn sonicator (Heat Systems, Farmingdate, N.Y.). Resulting cell homogenate was extracted with 4 ml of methanol, vortexed for 1 minute and centrifuged for 20 minutes at 3000 rpm. The samples were read by using HPLC. The concentration of drug in 10 cells was calculated using a standard calibration curve for drug spiked cells and were then treated similarly.

L3: Entry 109 of 128 File: USPT Oct 1, 1996

DOCUMENT-IDENTIFIER: US 5560923 A
TITLE: Method of encapsulating anthracycline in liposomes

Drawing Description Text (5):
FIG. 4 illustrates the reversal capacity of multidrug resistance by various treatments in LZ cells with liposome-encapsulated doxorubicin in LZ cells.

Detailed Description Text (30):
Study of the in vitro Drug Delivery Efficiency by Cardiolipin-Liposomes-Complexed Doxorubicin. Effect on Multidrug Resistance.

Detailed Description Text (31):
Resistance to major classes of cytotoxic drugs may emerge in tumor cells from patients treated by chemotherapy. Therefore, multidrug resistance may be one therapeutic obstacle in cancer treatment. It has been shown that liposome-encapsulated doxorubicin may modulate multidrug resistance in cancer cells. (A. R. Thierry, T. J. Jorgensen, D. Forst, I. A. Belli, A. Dritschilo, A. Rahman. Modulation of Multidrug Resistance in Chinese Hamster Cells by Liposome-encapsulated Doxorubicin. Cancer Comm. Vol. 1 pp. 311-316. (1989)). The capability to increase Doxorubicin activity in multidrug resistant cells was due to the use of a liposomal carrier. This capability was studied when cardiolipin-liposome-complexed doxorubicin. Thus, multidrug resistance reversal ability bore witness to the integrity or stability of the cardiolipin-liposome-doxorubicin complex.

Detailed Description Text (32):
Clonogenic assay was performed to evaluate modulation of multidrug resistance of free doxorubicin, cardiolipin-liposome-complexed doxorubicin and liposome-encapsulated doxorubicin in MCF-7/ADR and LZ cells which are resistant to doxorubicin. MCF-7/ADR and LZ cells are multidrug resistant cell lines originating from human breast cancer and Chinese hamster fibroblast,respectively.

Detailed Description Text (36):
FIG. 4 shows the reversal capacity of various treatments in the LZ cells. Generally, reversal capacity to multidrug resistance refers to the level at which drug resistance is overcome and is measured by the ratio of these amounts for free drug/liposome-encapsulated drug. For example, if 5 mg of free drug or 1 mg of liposome-encapsulated drug are required to overcome resistance to a drug, a reversal capacity of 5 is indicated. This would generally indicate that the liposome-encapsulated drug is five times as effective as the free drug in a drug-resistant host. Results demonstrate that increased concentrations of liposome in combination with Doxorubicin, for example substantially enhance the cytotoxic effect of the drug. As shown previously, liposome encapsulated doxorubicin seems to be approximately as cytotoxic as cardiolipin-liposome-complexed doxorubicin when liposome concentration (0.2 mg/ml) added to drug was equal to that present at equivalent drug concentration. For example, when 0.6 mg/ml and 1.0 mg/ml of liposomes (concentration corresponding to 90% and 50% survival for liposome cytotoxicity alone in LZ cells) are added to doxorubicin the reversal capacity of these treatments are 22 and 28 fold, respectively, showing a higher reversal capacity compared to liposome encapsulated doxorubicin (9-fold).

Detailed Description Text (37):
Evidence of the role of the complexed cardiolipin-liposome-doxorubicin on multidrug resistance is demonstrated in FIG. 2. LZ cells were exposed to different concentrations of cardiolipin-liposome-complexed doxorubicin corresponding to a liposome/doxorubicin weight ratio of 11. As previously observed, drug is nearly all complexed to the liposome when incubated at 37.degree. C. and the liposomal doxorubicin preparation exerts a cytotoxic effect higher than that of free drug. When vincristine (an alkaloid anticancer agent) is mixed in the same conditions as Doxorubicin to cardiolipin-containing liposomes, no association between drug and liposome was formed and no increase in drug cytotoxicity was observed. The results demonstrate that cardiolipin-liposome complexed specifically doxorubicin and that this association is responsible for the increase of cytotoxicity against multidrug resistant cells.

Previous Doc Next Doc Go to Doc#

L3: Entry 111 of 128 File: USPT Jun 13, 1995

DOCUMENT-IDENTIFIER: US 5424073 A
TITLE: Liposome encapsulated taxol and a method of using the same

Detailed Description Text (40):
Our studies demonstrate that liposome encapsulated taxol has significant capacity to overcome multidrug resistance. These studies were performed in HL 60/VCR cells which are promyelocytic human leukemia cells and are derived from parent HL-60 cells and are made resistant to vincristine demonstrating multidrug resistance phenotype. The HL-60 VCR cells were grown in media at 37.degree. C. with 5% CO.

Detailed Description Text (42):
The survival curves of HL-60/VCR cells after exposure to free taxol and liposome encapsulated taxol are presented in FIG. 5. The IC.sub.50 for free taxol in HL-60/VCR is 2.43 .mu.g/ml demonstrating this cell line is 1280 fold resistant than the parent HL-60 cell line. However, the IC.sub.50 for liposomal taxol is only 0.37 .mu.g/ml. This demonstrates that liposomal taxol sensitizes 7 fold the HL-60/VCR as compared to free taxol. Hence, it is apparent that liposome encapsulated taxol modulates drug resistance in the multidrug resistance phonotype which potentially would be a major advantage in a clinical situation where a large population of the patients fail to respond to chemotherapy because of multidrug resistance.

Detailed Description Text (44):
The cellular content of taxol was determined by High Pressure Liquid Chromatography. In brief, the multidrug resistance HL-60/VCR exponential growth phase cells were incubated in 100 mm petri dishes with drug containing medium at 37.degree. C. containing either free taxol or taxol encapsulated in liposomes. The cells were treated for 1 to 4 hours with either of the drug and were then centrifuged and rinsed twice with PBS. The cells were suspended in cold PBS and counted, and centrifuged again. The cell pellets were suspended in 0.5 ml of 2% SDS solution and then sonicated for 5 minutes in a cup-horn sonicator (Heat Systems, Farmingdate, N.Y.). Resulting cell homogenate was extracted with 4 ml of methanol, vortexed for 1 minute and centrifuged for 20 minutes at 3000 rpm. The samples were read by using HPLC. The concentration of drug in 10 cells was calculated using a standard calibration curve for drug spiked cells and were then treated similarly.

Refine Search

Search Results -

Terms	Documents
liposome same (multidrug adj1 resist$)	128

Database:
US Pre-Grant Publication Full-Text Database
US Patents Full-Text Database
US OCR Full-Text Database
EPO Abstracts Database
JPO Abstracts Database
Derwent World Patents Index
IBM Technical Disclosure Bulletins

Search: L3

Refine Search | Recall Text | Clear | Interrupt

Search History

DATE: Wednesday, March 07, 2007 **Purge Queries** Printable Copy Create Case

Set Name side by side	Query	Hit Count	Set Name result set
DB=PGPB,USPT,USOC,EPAB,JPAB,DWPI,TDBD; PLUR=YES; OP=OR			
L3	liposome same (multidrug adj1 resist$)	128	L3
L2	mitomycin same (multidrug adj1 resist$)	58	L2
L1	mitomycin same multidrug	67	L1

END OF SEARCH HISTORY

First Hit Fwd Refs Previous Doc Next Doc Go to Doc#

Generate Collection Print

L3: Entry 86 of 128 File: USPT Feb 22, 2000

DOCUMENT-IDENTIFIER: US 6028054 A
** See image for Certificate of Correction **
TITLE: Method for increasing bioavailability of oral pharmaceutical compositions

Brief Summary Text (68):
Thierry, A. R., D. Vige, S. S. Coughlin, J. A. Belli, A. Dritschilo, and A. Rahman. "Modulation of doxorubicin resistance in multidrug-resistant cells by liposomes." FASEB J (6 1993): 572-9.

Brief Summary Text (70):
Warren, L., J.-C. Jardiller, A. Malarska, and M.-G. Akeli: "Increased accumulation of drugs in multidrug-resistance cells induced by liposomes" Cancer Research 52:3241 (1992).

Other Reference Publication (62):
Thierry et al., "Modulation of Doxorubicin Resistance in Multidrug-Resistant Cell by Liposomes," FASEB J., 6:572-579 (1993).

Other Reference Publication (64):
Warren et al., "Increased Accumulation of Drugs in Multidrug-Resistant Cell Induced by Liposomes," Cancer Research, 52:3241-3245 (1992).

Previous Doc Next Doc Go to Doc#